Amendments to the Specification

Please replace the paragraph at page 22, line 22 to page 23, line 3 with the following paragraph:

Thus, the nucleic acid molecules of the present invention typically comprise either a homologous or heterologous promoter sequence and other suitable control sequences. These other control sequences may comprise a terminator and/or translation initiation sequence (e.g. GCCACCATGG (SEQ ID NO: 1) or GCCCCCATGG (SEQ ID NO: 2)) and/or translational stop codon (e.g. TAA, TAG or TGA) and/or polyadenylation signal and/or a RNA pause site. In addition, native or heterologous enhancer sequences for the promoter sequence may be present. Once constructed, the nucleic acid molecules can be administered using standard gene delivery protocols. Methods for gene delivery are known in the art. See, e.g., U.S. Patent Nos. 5,399,346, 5,580,859, 5,589,466. Genes can be delivered either directly to a subject or, alternatively, delivered *ex vivo*, to cells derived from the subject and the cells reimplanted in the subject.

Please replace the paragraph at page 23, line 6 to page 24, line 11 with the following paragraph:

The second component of the novel compositions of the present invention is the adjuvant component which can comprise any suitable adjuvant or combination of adjuvants. For example, suitable adjuvants include, without limitation, adjuvants formed from aluminum salts (alum), such as aluminum hydroxide, aluminum phosphate, aluminum sulfate, etc; oil-in-water and water-in-oil emulsion formulations, such as Complete Freunds Adjuvants (CFA) and Incomplete Freunds Adjuvant (IFA); mineral gels; block copolymers; Avridine™ lipid-amine; SEAM62; adjuvants formed from bacterial cell wall components such as adjuvants including lipopolysaccharides (e.g., lipid A or monophosphoryl lipid A (MPL), Imoto et al. (1985) *Tet. Lett.* 26:1545-1548), trehalose dimycolate (TDM), and cell wall skeleton (CWS); heat shock protein or derivatives thereof; adjuvants derived from ADP-ribosylating bacterial toxins, including diphtheria toxin (DT), pertussis toxin (PT), cholera toxin (CT), the *E. coli* heat-labile toxins (LT1 and LT2), *Pseudomonas* endotoxin A, *Pseudomonas* exotoxin S, *B.*

cereus exoenzyme, *B. sphaericus* toxin, *C. botulinum* C2 and C3 toxins, *C. limosum* exoenzyme, as well as toxins from *C. perfringens*, *C. spiriforma* and *C. difficile*, *Staphylococcus aureus* EDIN, and ADP-ribosylating bacterial toxin mutants such as CRM_{197}, a non-toxic diphtheria toxin mutant (see, e.g., Bixler et al. (1989) *Adv. Exp. Med. Biol.* 251:175; and Constantino et al. (1992) *Vaccine*); saponin adjuvants such as Quil A (U.S. Pat. No. 5,057,540), or particles generated from saponins such as ISCOMs (immunostimulating complexes); chemokines and cytokines, such as interleukins (e.g., IL-1, IL-2, IL-4, IL-5, IL-6, IL-7, IL-8, IL-12, etc.), interferons (e.g., gama interferon), macrophage colony stimulating factor (M-CSF), tumor necrosis factor (TNF), defensins 1 or 2, RANTES, MIP1- and MIP-2, etc; muramyl peptides such as N-acetyl-muramyl-L-threonyl-D-isoglutamine (thr-MDP), N–acetyl-normuramyl-^{L}-alanyl-^{D}-isoglutamine (nor-MDP), N-acetylmuramyl-^{L}-alanyl-^{D}-isoglutaminyl-^{L}-alanine-2- (1' -2' -dipalmitoyl-*sn*-glycero-3 huydroxyphosphoryloxy)-ethylamine (MTP-PE) etc.; adjuvants derived from the CpG family of molecules, CpG dinucleotides and synthetic oligonucleotides which comprise CpG motifs (see, e.g., Krieg et al. *Nature* (1995) 374:546, Medzhitov et al. (1997) *Curr. Opin. Immunol.* 9:4-9, and Davis et al. *J. Immunol.* (1998) 160:870-876) such as TCCATGACGTTCCTGATGCT (SEQ ID NO: 3) and ATC *C. limosum* exoenzyme GACTCTCGAGCGTTCTC (SEQ ID NO: 4); and synthetic adjuvants such as PCPP (Poly[di(carboxylatophenoxy)phosphazene) (Payne et al. *Vaccines* (1998) 16:92-98). Such adjuvants are commercially available from a number of distributors such as Accurate Chemicals; Ribi Immunechemicals, Hamilton, MT; GIBCO; Sigma, St. Louis, MO.

Please replace the paragraph at page 43, lines 14 to 26 with the following paragraph:

The NP DNA vector contained a sequence encoding the influenza NP antigen (from the PR8 strain) and is described in Pertmer et al. (1995) *Vaccine* 13:1427-1430. All gold beads were loaded as described above at a bead loading rate of 0.5 mg Au/target and a DNA loading rate of 2.0 μg/mg Au. Administration with the PowderJect™ XR device was to two target sites at 400 psi operating pressure, and was a prime-only experiment. After one month, mice were sacrificed and blood and spleen samples collected for analysis as also described above. For the ELISPOT assay, the peptide used to stimulate the CTL precursors was

TYQRTRALV (SEQ ID NO: 5) (at 10^{-8}M) and the lysed influenza PR8 virus was used at 5 μg/mL for the Th stimulation. For the chromium analysis, the peptide used to stimulate the responders was TYQRTRALV (SEQ ID NO: 5) (at 10^{-8}M); and the peptide used to pulse the targets was TYQRTRALV (SEQ ID NO: 5) (at $10^{-5.5}$M). For the ELISA, the capture antigen was PR8 NP antigen (Sprafas) at 1 mg/mL.

Please replace the paragraph at page 44, line 27 to page 45, line 11 with the following paragraph:

Here again, the NP DNA vector contained a sequence encoding the influenza NP antigen (from the PR8 strain) and is described in Pertmer et al. (1995) *Vaccine* 13:1427-1430. All gold beads were loaded as described above at a bead loading rate of 0.5 mg Au/target and a DNA loading rate of 2.0 μg/mg Au. Administration with the PowderJect™ XR device was to two target sites at 400 psi operating pressure, and was a prime-only experiment. Vaccination was only given once, and after one month, mice were sacrificed and blood and spleen samples collected for analysis as also described above. For the ELISPOT assay, the peptide used to stimulate the CTL precursors was TYQRTRALV (SEQ ID NO: 5) (at 10^{-8} M) and the lysed influenza PR8 virus was used at 5 μg/mL for the Th stimulation. For the chromium analysis, the peptide used to stimulate the responders was TYQRTRALV (SEQ ID NO: 5) (at 10^{-8}M); and the peptide used to pulse the targets was TYQRTRALV (SEQ ID NO: 5) (at $10^{-5.5}$M). For the ELISA, the capture antigen was PR8 NP antigen (Sprafas) at 1 mg/mL.

Please replace the paragraph at page 46, lines 17 to 28 with the following paragraph:

The HIV gp120 DNA vector contained a sequence encoding the HIV gp120 antigen (from the LAI strain) and is described in Heydenburg et al. (1994) *AIDS Res. and Hum. Retroviruses* 10:1433-1441. All gold beads were loaded as described above at a bead loading rate of 0.5 mg Au/target and a DNA loading rate of 2.0 μg/mg Au. Administration with the PowderJect™ XR device was to two target sites at 400 psi operating pressure. Varying numbers of vaccinations were carried out (some having one boost, others having three boosts), with vaccination administered every four weeks. Sacrifice of all mice was at week

14 of the study, and spleen samples were collected for analysis as described above. For the ELISPOT assay, the peptide used to stimulate the CTL precursors was RGPGRAFVTI (SEQ ID NO: 6) (at 10^{-7}M). For the chromium analysis, the peptide used to stimulate the CTL responders and pulse the targets was RGPGRAFVTI (SEQ ID NO: 6) (at 10^{-7}M).

Please replace the paragraph at page 47, line 30 to page 49, line 3 with the following paragraph:

The HbsAg DNA vector contained a sequence encoding the HBV surface antigen is termed pWRG7128. The plasmid pWRG7128 contains, in addition to suitable control elements, a sequence encoding the hepatitis B surface antigen (HbsSAg) which is under the transcriptional control of a cytomegalovirus (CMV) promoter, and has been shown to produce HbsAg particles upon transfection into most cell types. The pWRG7128 plasmid was constructed as follows. A cloning vector pWRG7077 (Schmaljohn et al. (1997) *J. Virol.* 71:9563-9569) was prepared to accept a HbsAg coding sequence by digesting the vector to completion with *Bam*H1, followed by a partial digest with *Hind*3. After blunting the 5' overhangs by treatment with Klenow fragemnt and deoxyribonucleotides, the 4.3 kB vector fragment was isolated. The 1.35 kB HbsAg insert fragment (containing the untranslated pre-S2 sequence, the 226 amino acid HbsAg coding sequence of the *adw* strain, and the HBV enhancer element) was excised from plasmid pAM6 (ATCC, Rockford, MD) by digesting with *Bam*H1. After blunt-ending by treatment with the Klenow fragment and deoxyribonucleotides, the fragment was isolated and ligated into the 4.3 kB vector fragment described above. The resulting recombinants were screened for proper orientation of the insert and a correct isolate was identified and designated as an intermediate plasmid (pWRG7074). In order to remove the start of the codon of the X protein (present at the 3' end of the pAM6 1.35 kB insert), a 4.86 kB vector fragment was isolated from the pWRG7074 plasmid by digesting with *Bgl*2, blunt-ending with the Klenow fragment and deoxyribonucleotides, and then digesting with *Bst*X1. Next, a 754 bp insert fragment was isolated from the pWRG7074 construct by digestion with *Nco*1, treating with mung bean nuclease, and digesting with *Bst*X1. The resulting vector and insert fragments were then ligated together to form the clinical plasmid pWRG7128. All gold beads were loaded as

described above at a bead loading rate of 0.5 mg Au/target and a DNA loading rate of 2.0 μg/mg Au. Administration with the PowderJect™ XR device was to two target sites at 400 psi operating pressure. Vaccination was only given once, and after one month, mice were sacrificed and spleen samples collected for chromium release (specific lysis) analysis as described above. For the ELISPOT assay, the peptide used to stimulate the CTL precursors was IPQSLDSWWTSL (SEQ ID NO: 7) (at 10^{-6}M). For the chromium analysis, a P815 cell line expressing the HBV surface antigen was used instead of peptide for stimulation of responders (at 40,000/well) and for effectors (at 30,000/well).